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Central somatosensory changes and altered muscle synergies in subjects with anterior cruciate ligament deficiency

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Abstract

To clarify the mechanisms of compensation in subjects with anterior cruciate ligament deficiency (ACL-D), we examined proprioception, quadriceps strength, somatosensory evoked potentials (SEPs) and muscle responses during gait in varied combinations of speed and incline. Seventeen subjects with ACL-D were grouped by functional level and report of giving way. Seven subjects without ACL-D served as a control sample for muscle response measures. ACL-D subjects with quadriceps weakness experienced giving way and could not resume sport activities. Those without weakness fell into one of two groups: (1) copers, who had full return to previous activity and no giving way despite proprioceptive loss and altered SEPs, and (2) adapters, who were unable to return to previous activity level and experienced giving way despite neither proprioceptive loss or altered SEPs. The unique muscle pattern in copers during inclined fast walking included larger and earlier hamstring activation. These results suggest that in individuals with ACL-D without a strength deficit, altered SEPs and altered neuromuscular patterns are the factors that enable resumption of pre-injury functional levels. Loss of proprioception may drive the central changes, which in turn drives the development of altered muscle patterns.

Keywords: Proprioception; Somatosensory evoked potentials; Compensation; Anterior cruciate ligament

1. Introduction

Although, the role of somatosensory information in the control of gait is well documented [1], the mechanisms of compensation in individuals with peripheral somatosensory loss, such as those with complete tear of the anterior cruciate ligament (ACL-D), have not been clearly delineated. Some patients with ACL-D recover and return to sport (copers) [2–5] while others complain of instability and 'giving way' with basic activities (non-copers) [2–4]. Reports of the mechanisms responsible for recovery in copers, or the lack thereof in non-copers, have been conflicting [6–10]. Research has confirmed that the passive knee laxity in individuals with ACL-D is unrelated to the ability to return to sports [5]. It has been proposed that the lack of proprioception or central nervous system changes in response to proprioceptive loss may determine the different outcomes [11]. Inconclusive reports may be due to the divergent methodologies used and the lack of concurrent measurement of function, strength, proprioception and neuromuscular responses that is needed to answer this question.

1.1. Mechanisms of compensation

Proposed mechanisms for compensation following ACL-D include: (1) maintenance of proprioception at the knee as measured by threshold to detection of passive motion (TDPM) [8,10]; (2) activation of a capsular-hamstring reflex due to increased mechanical laxity at the joint [6,7] and (3) modulation of central programming [9,11]. Mechanoreceptors within the ACL contribute to the compilation of limb position information from numerous receptors [12,13] that enables the dynamic postural control

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needed for gait [1]. Reportedly, the giving way is related to higher thresholds on TDPM testing [8,10]. Johansson et al. [14] suggested that as a consequence of proprioceptive loss, the ensemble sensory feedback and existing motor program is disrupted, resulting in the 'giving way' reported. However, conclusions must be guarded. Weakness alone can result in giving way, and strength was not measured in these studies. Furthermore, to relate proprioceptive loss to alteration or lack of muscle response and consequent giving way requires measurement of neuromuscular output in those with and without proprioceptive loss, which was not done in these studies.

Altered neuromuscular patterns have been reported in subjects with ACL-D during resisted knee extension and gait [6,15,16]. Investigators [6,16] concluded that poor functional outcome was due to peripherally mediated reflex stiffness at the knee, with changes in timing, not amplitude of muscle responses. However, neither proprioception nor strength was measured in any of these studies. Di Fabio et al. [9] postulated that the altered synergies may represent central reorganization.

Di Fabio et al. [9] reported earlier and less variable response of hamstrings in the affected ACL-D limb, as compared to the unimpaired limb during an external platform perturbation, and dominance of hamstring, as compared to quadriceps, activity during unilateral stance on the ACL-D limb. These authors [9] concluded that the generalized increase in gain of hamstring response was due to increased input from the joint capsule of the involved limb, with secondary changes in central nervous system activity and the output generated. However, without concurrent measurement of proprioception and central changes, these conclusions may be overstated.

Somatosensory evoked potential (SEP) testing yields electrical recordings representative of activity in peripheral and central neural pathways in response to external stimuli. Using this test, investigators have demonstrated evoked potentials in response to stimulation of an intact ACL [17], and absence of the P27 potential in ACL-D subjects with proprioceptive loss [11]. Valeriani et al. [11] postulated that loss of the P27 potential represents central nervous system (CNS) reorganization. Although tempting to conclude that functional deficits in those with ACL-D are due to loss of proprioception and central changes, measures of function, leg strength, and neuromuscular activation patterns were not obtained.

To address the question of compensation, or the lack thereof, and to identify the mechanisms involved requires concurrent measurement of functional status, neuromuscular response patterns, proprioception and SEPs. The purpose of this study was to obtain these measures and subjective report of function in subjects with ACL-D, and to compare response measures from involved and un-involved limbs. Identification of the mechanisms enabling compensation and recovery may assist in the development of optimal interventions for individuals with ACL-D.

2. Methods

We compared proprioception and SEPs between groups of individuals with ACL-D and compared muscle synergies during gait between groups with ACL-D as well as to a normative sample.

2.1. Subjects

Seven males and ten females of 23–50 years of age (mean age 34.5, s.d. 8.6 years) with ACL-D who never had surgical repair agreed to participate. Seven 23- to 34-year-old individuals (six females and one male; mean age 27, s.d. 4.1 years) without ACL-D participated as a control sample for the treadmill walking test. Exclusion criteria for all subjects were: (1) history of any vestibular dysfunction; (2) uncorrected vision problems; (3) history of orthopedic lower extremity injury and (4) history of neurological disease or injury. Informed consent, as approved by the University of Miami Medical Sciences Review Committee, was obtained prior to testing.

ACL-D was confirmed by arthroscopy or MRI. All subjects were at least 2 months post-injury, had no swelling, pain, limiting gait deviation or limits in passive knee range of motion. Each subject completed a questionnaire regarding medical history and activity level and in the case of subjects with ACL-D, incidence of giving way and ability to return to cutting and pivoting sport activities. Based on reports of functional level and giving way, subjects were placed into one of two groups: (1) noncopers, who complained of numerous incidents of giving way during typical daily activities and difficulty with any sport activity, and (2) copers, who never experienced giving way and had a full return to sport activity, to include cutting and pivoting tasks.

2.2. Experimental procedures

2.2.1. Strength testing: maximum voluntary isometric contraction of the quadriceps

The maximum voluntary isometric contraction (MVIC) force of the subjects' quadriceps femoris muscle was determined by using a burst-superimposition method on both lower extremities [18]. The subject was seated on an isokinetic dynamometer [19] with the ankle secured to the dynamometer force arm and the hip and knee placed at 90° (resting position). Self-adhesive stimulating electrodes were applied to the proximal vastus lateralis and the vastus medialis muscles. A supramaximal burst of electrical stimulation (100 pulses per second, 600 μs pulse duration, 10 pulse tetanic train) [20] was superimposed on a maximal voluntary contraction, with force recorded by the dynamometer. Three trials were completed on each limb to obtain the peak MVIC. Relative strength or quadriceps index (QI) of the involved leg was calculated from peak MVIC of each limb (QI = (involved limb peak MVIC/uninvolved limb peak